SPECTRUM OF INTRACARDIAC CLOT

Dr. Anish Khan

Resident

Department of Cardiology

- Case-1
- 47 years old male known case of RVHD/SEVERE MS/MILD MR/ MODERATE AR/ MILD AS/MILD TR/ MODERATE PAH

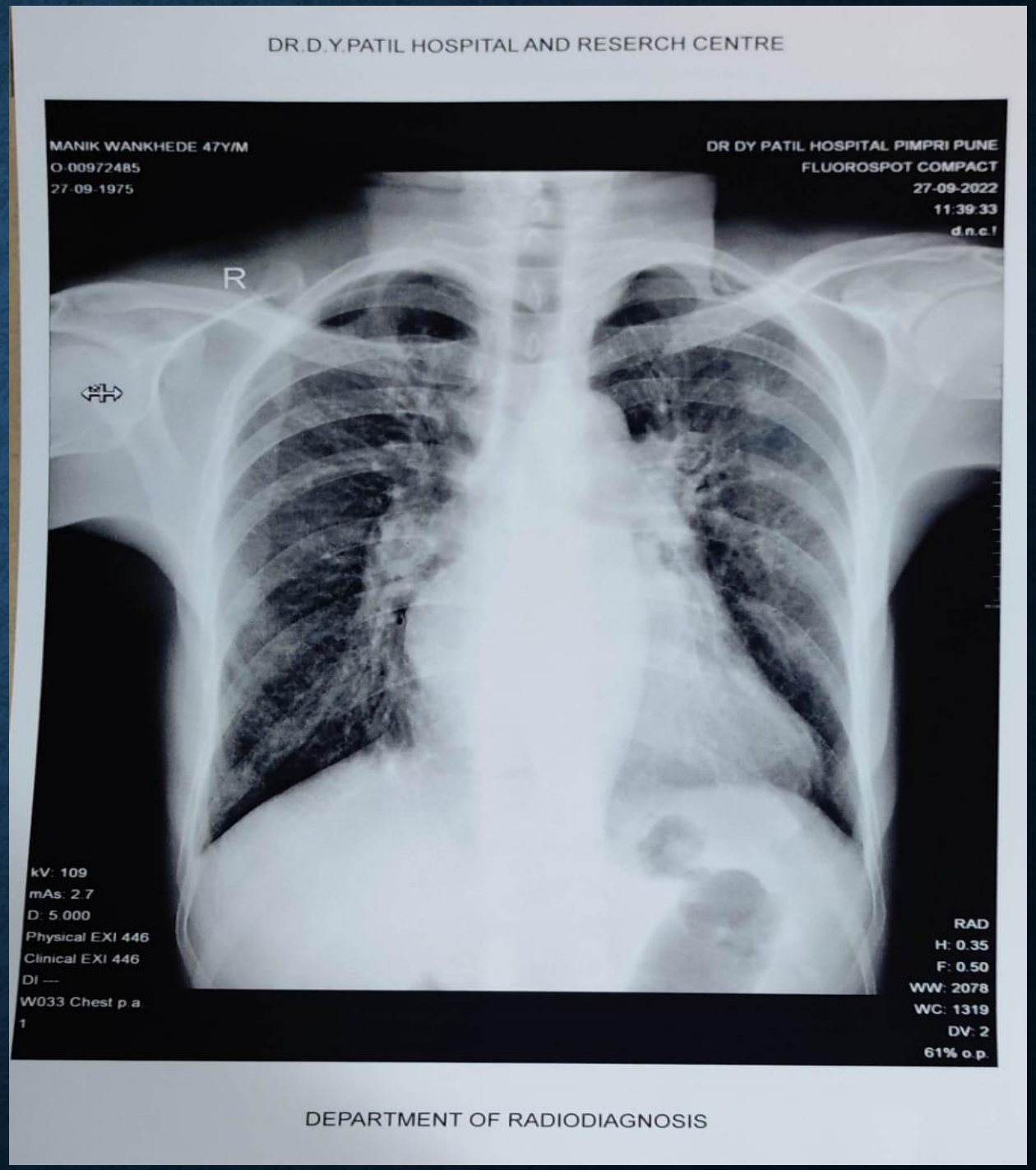
SYSTEMIC EXAMINATION:-

INSPECTION:-

- APICAL IMPULSE-5TH ICS LATERAL TO MID CLAVICULAR LINE
- PALPATION:-
- APICAL IMPULSE -TAPPING AND SITE IS CONFIRMED

CXR:-

- **STRAIGHTNING OF LEFT HEART BOARDER**
- **DILATED LA**
- **WIDENING OF CARINA**
- **LV APEX**



MANIK WANKHEDE
47 Years Male

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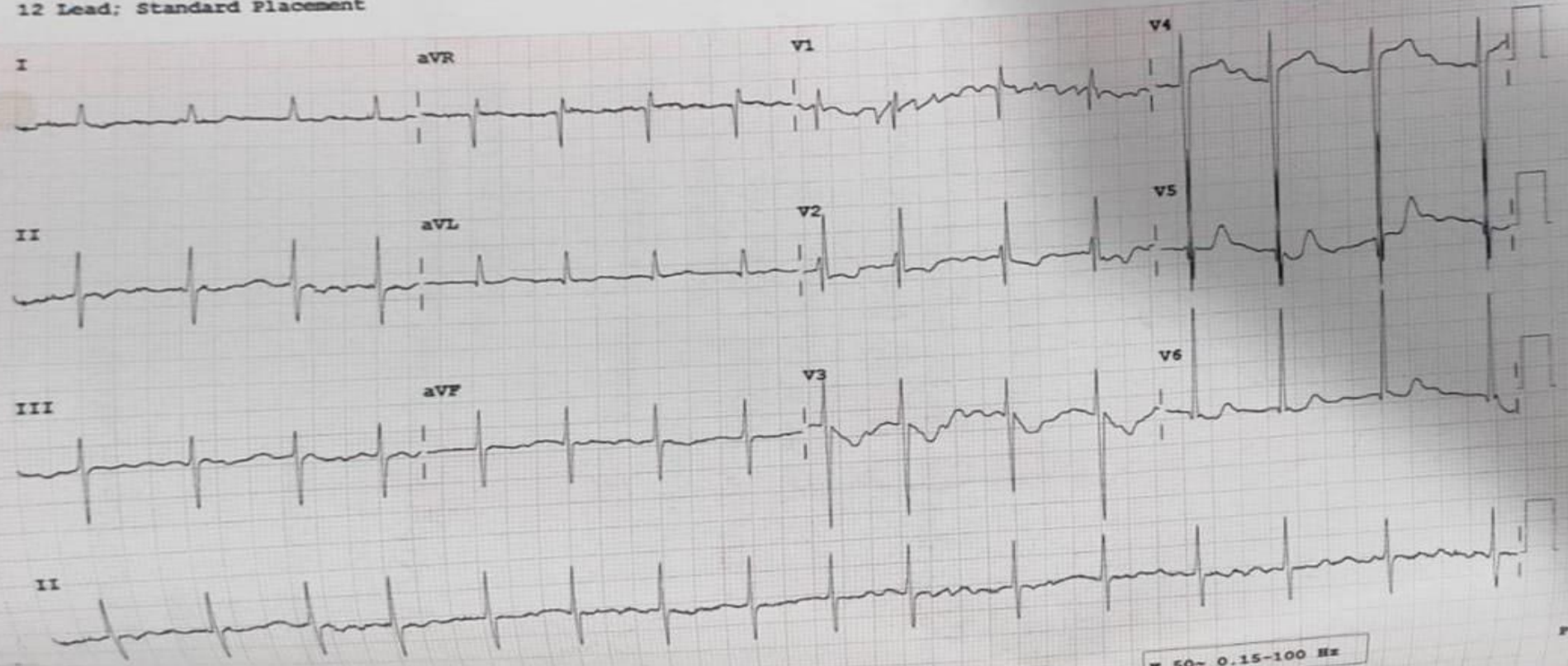
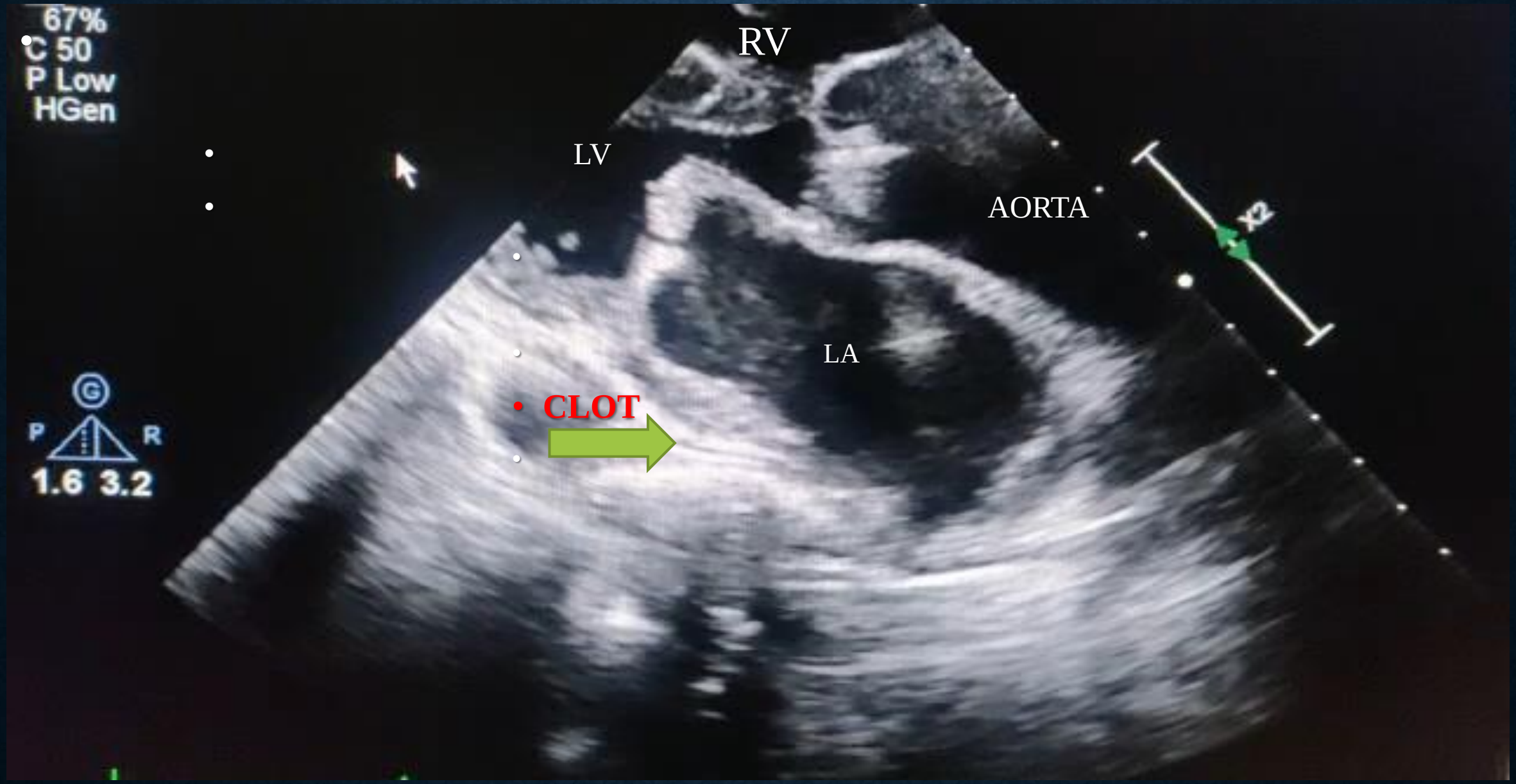
(DR.D.Y.PATIL HOSPITAL)
DR.D.Y.PATIL HOSPITAL
Medicine OPD

6

12 Lead; Standard Placement



- **2D ECHO:-**
- DILATED LA,RA,RV
- LA clot with spontaneous echo contrast of grade-III noted in LA
- Normal LV systolic function, LVEF-60%
- SEVERE MS,MILD MR,MVA BY PLANNIMETRY-0.9 CM2,MVPG/MG-19/11 MMHG
- Moderate AR, Mild As AVPG/MG-34/18, AV PHT-395 msec, AVS
- MOD TR,MOD PAH RVSP-55MMHG
- IVC-DILATED <50% COLLAPSING WITH RESPIRATION


67%
C 50
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HGen
RV
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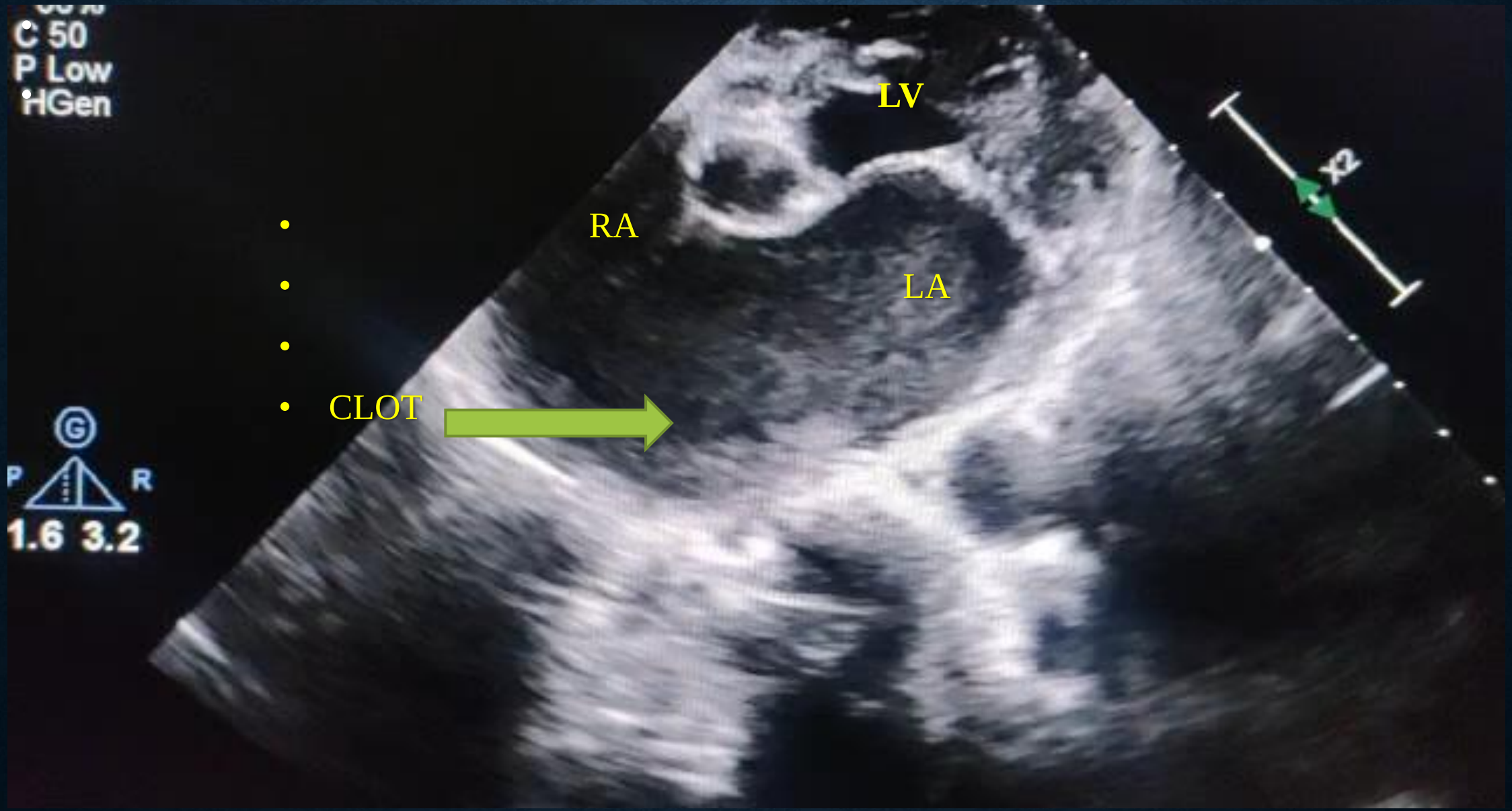
AORTA
LA
CLOT
1.6 3.2


C 50
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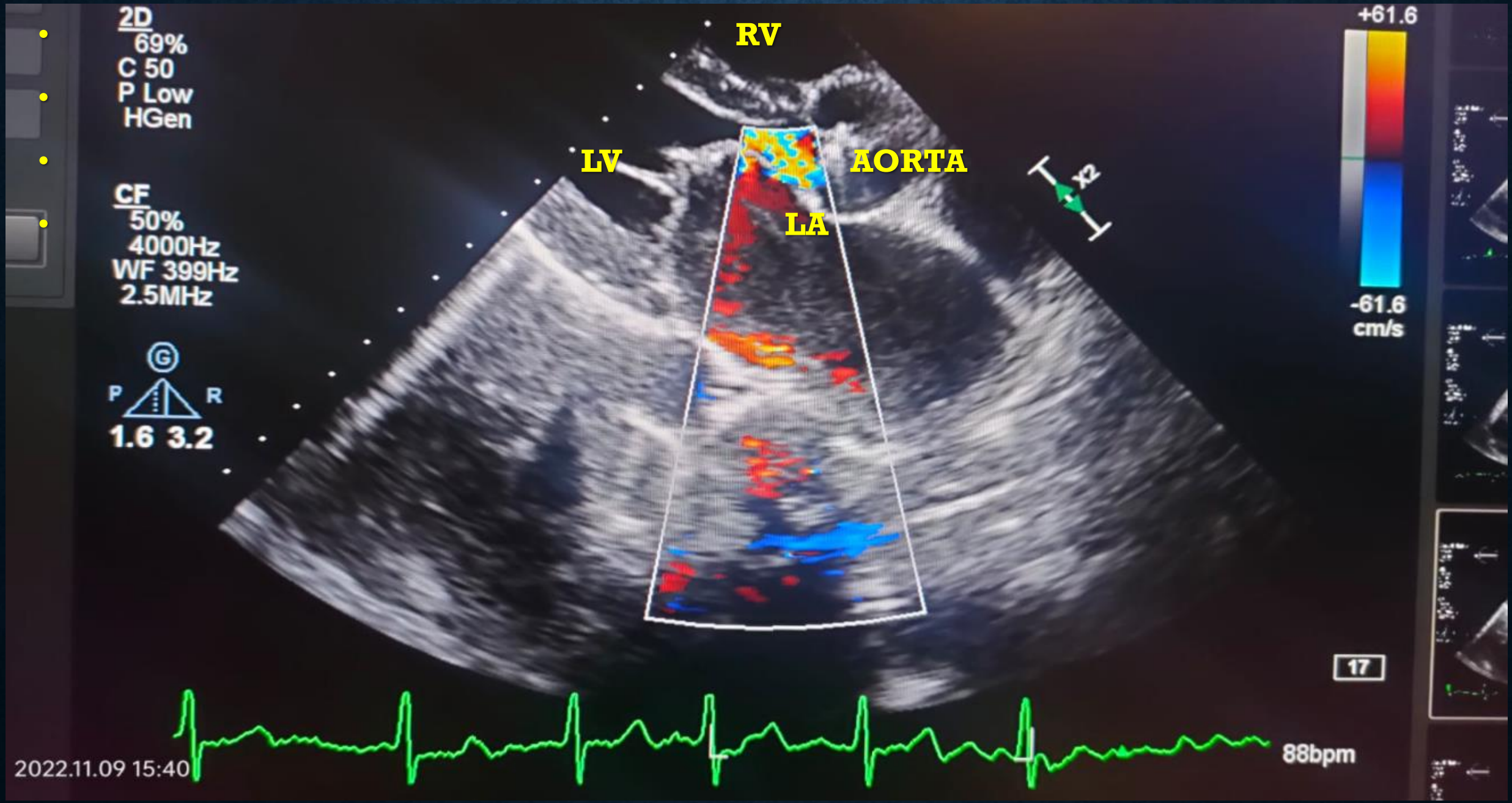
1.6 3.2


2D
69%
C 50
P Low
HGen
CF
50%
4000Hz
WF 399Hz
2.5MHz
1.6 3.2
RV
LV
AORTA
LA
+61.6
-61.6
cm/s
17
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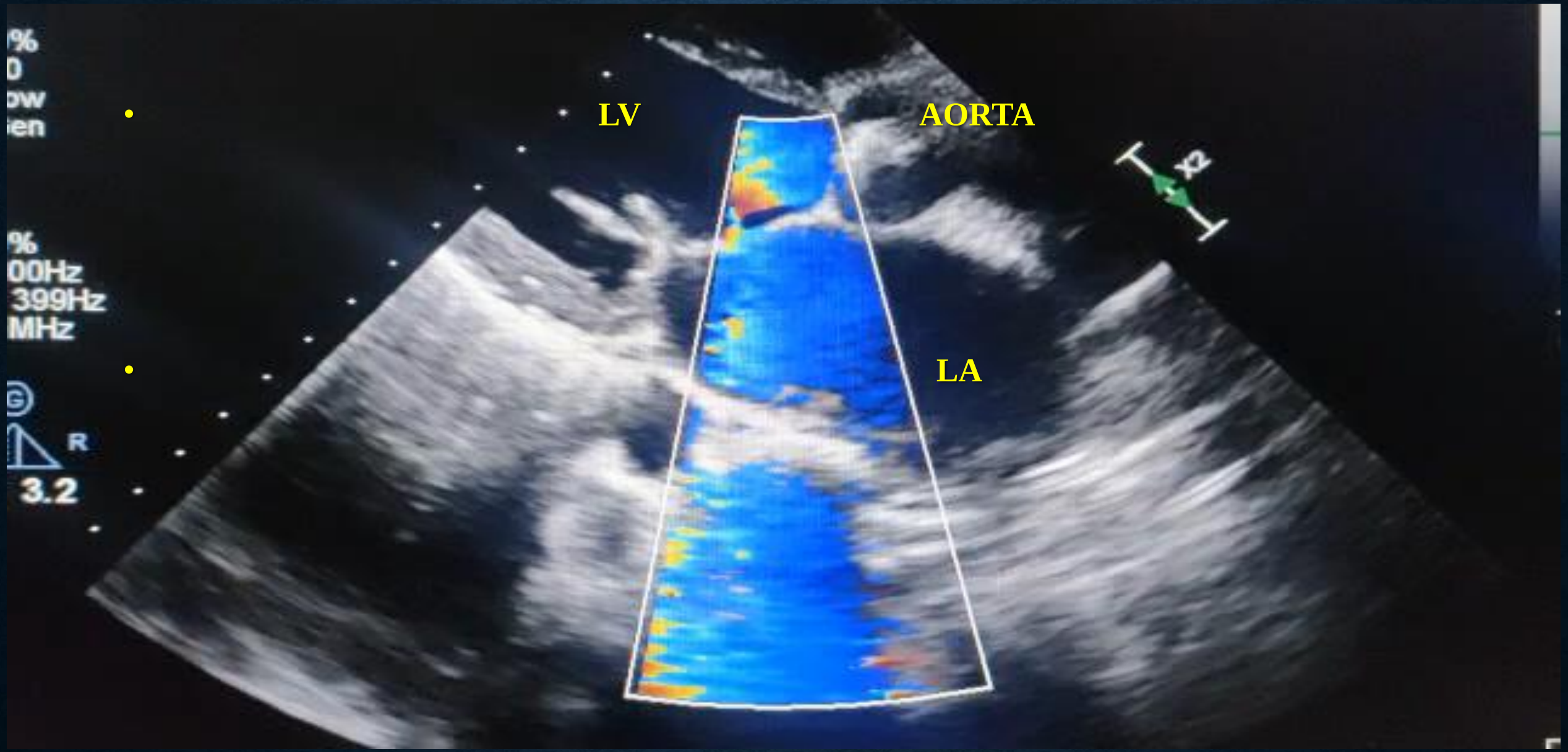
88bpm


LV
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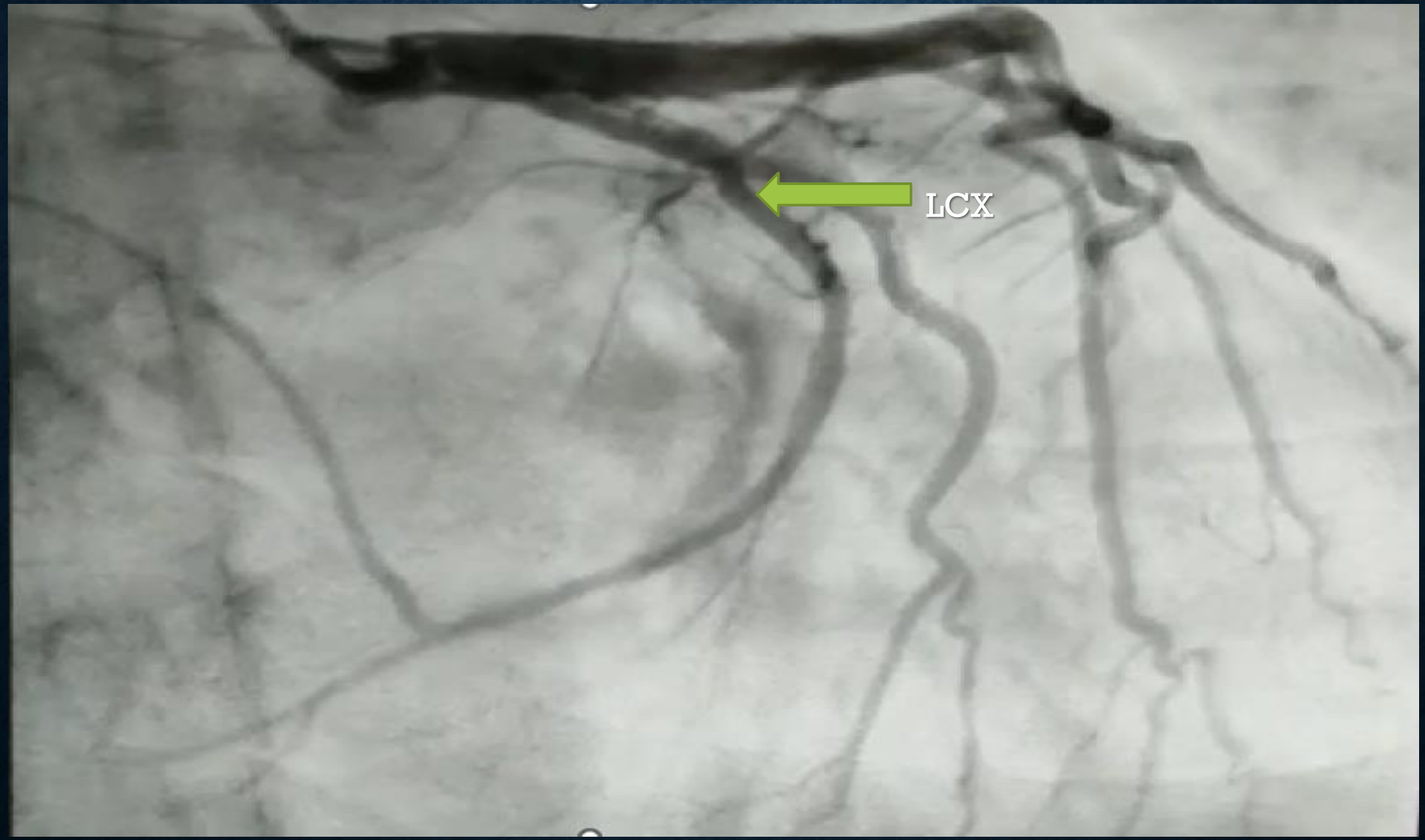
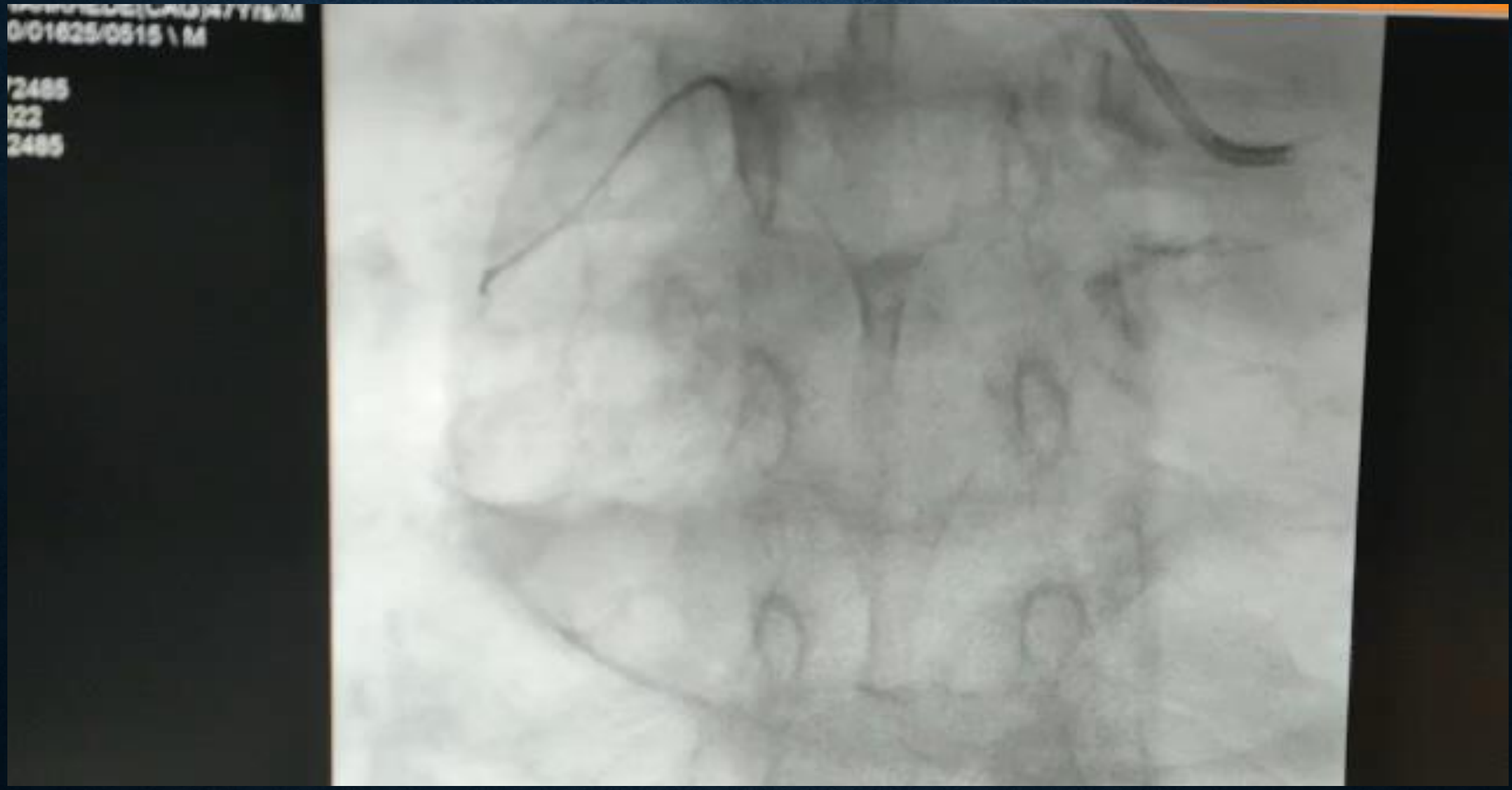
CAG



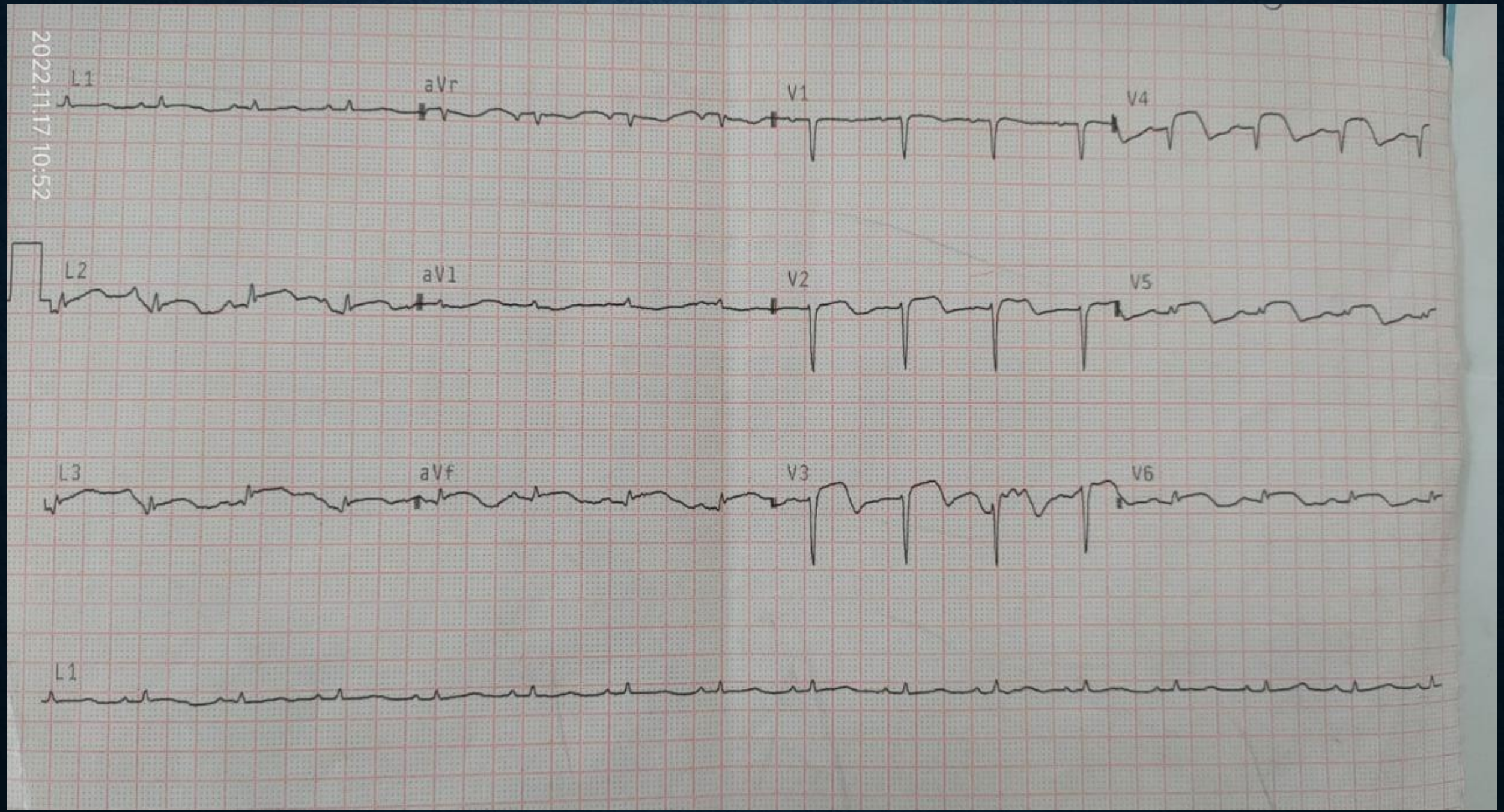
CAG



TREATMENT

- If there is a isolated LA clot in a patient with MS with pliable valve the treatment of choice is to give anticoagulation for three months and look for the resolution of the clot.
- If the clot resolves than we an go ahead with BMV.
- In this case there was associated Mitral & Aortic valve diseases and mitral valve was not pliable and calcified , so we advised DVR with removal of LA clot.(Neovascularization was another criteria for removal as it is not going to dissolve with anticoagulation).
- Patients with rheumatic mitral stenosis with atrial fibrillation should receive oral anticoagulation with a vitamin K antagonist to prevent LA/LAA clot formation.
- Patients with nonvalvular atrial fibrillation with CHA2DS2-VASc score>= 1in men and >=2 in women should receive VKA or NOAC for prevention of LA thrombus and stroke.

- Case-2
- 46years old female known case of CAD-Recent AWMI/Moderate LV dysfunction , LVEF-40-45%, SVD-LAD(Distal total thrombotic occlusion)
- C/O:-
- Pain and tenderness over the dorsum of right leg.
- General Examination:-
- Height-160 cm
- Weight- 65 Kg
- BMI-25.39/Kg/m2
- BP- 130/80, PR- 80 bpm, (Right sided Popliteal, Dorsalis pedis, Posterior tibialis Pulses absent) Spo2-98% in R.A., Afebrile
- Systemic Examination:-
- R/S:- BSBE, B/L-VBS-+, CVS-S1 & S2-Normal, No added sounds , No murmur heard, CNS- conscious , others-normal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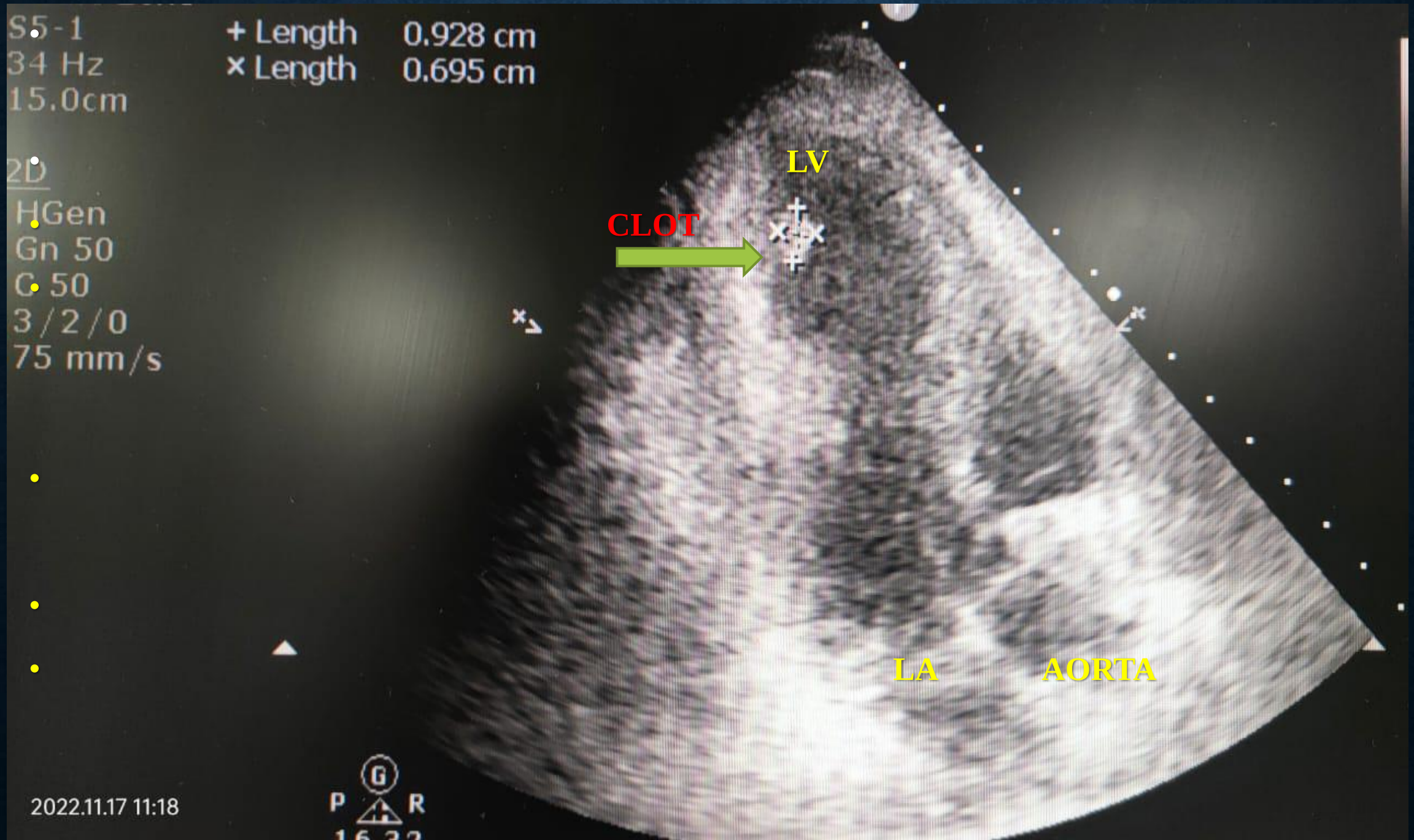
L1
aVr
V1
V4
L2
aV1
V2
V5
L3
aVf
V3
V6
L1


S5-1
34 Hz
15.0cm
2D
HGen
Gn 50
C 50
3/2/0
75 mm/s
+ Length 0.928 cm
× Length 0.695 cm
LV
CLOT
LA
AORTA
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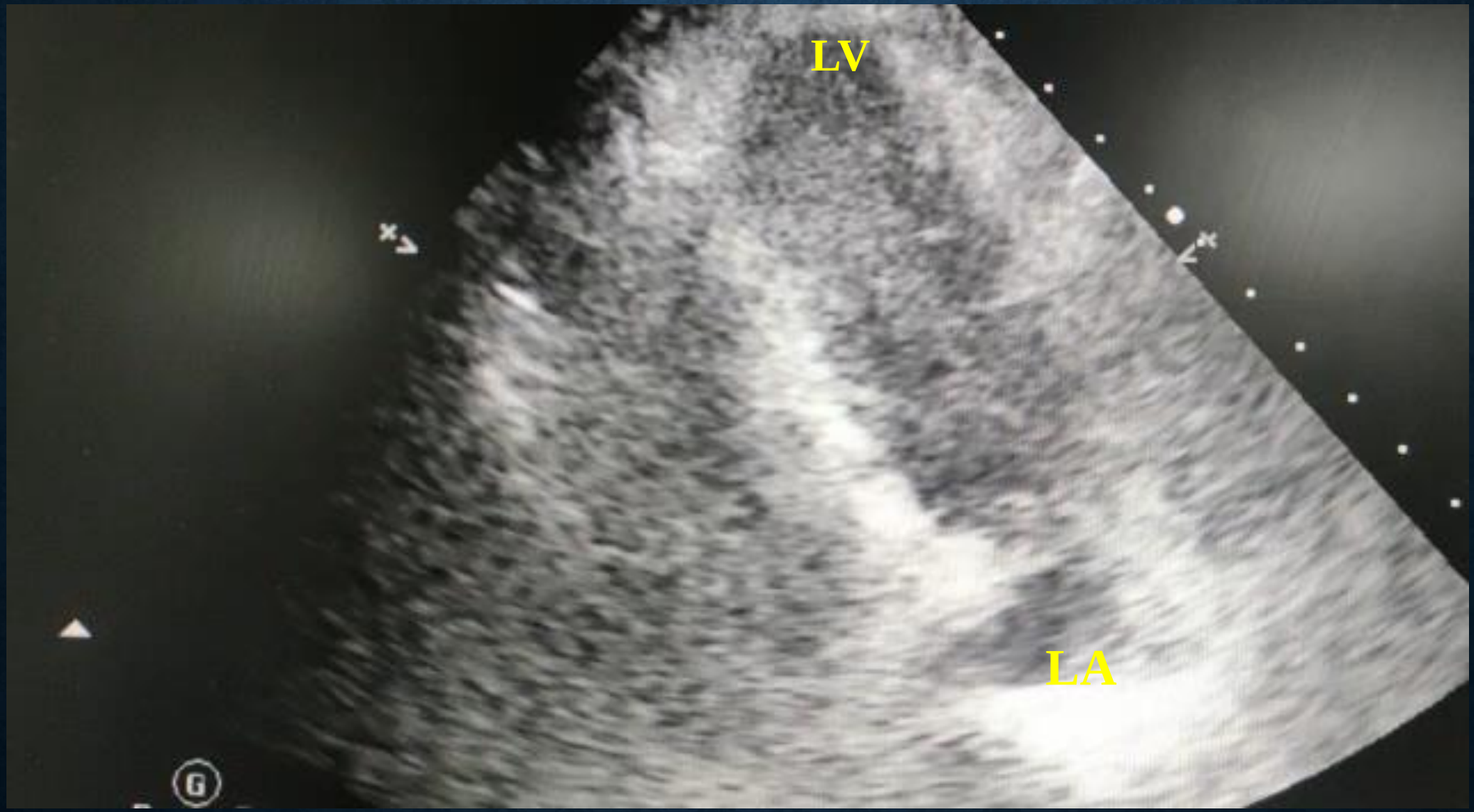
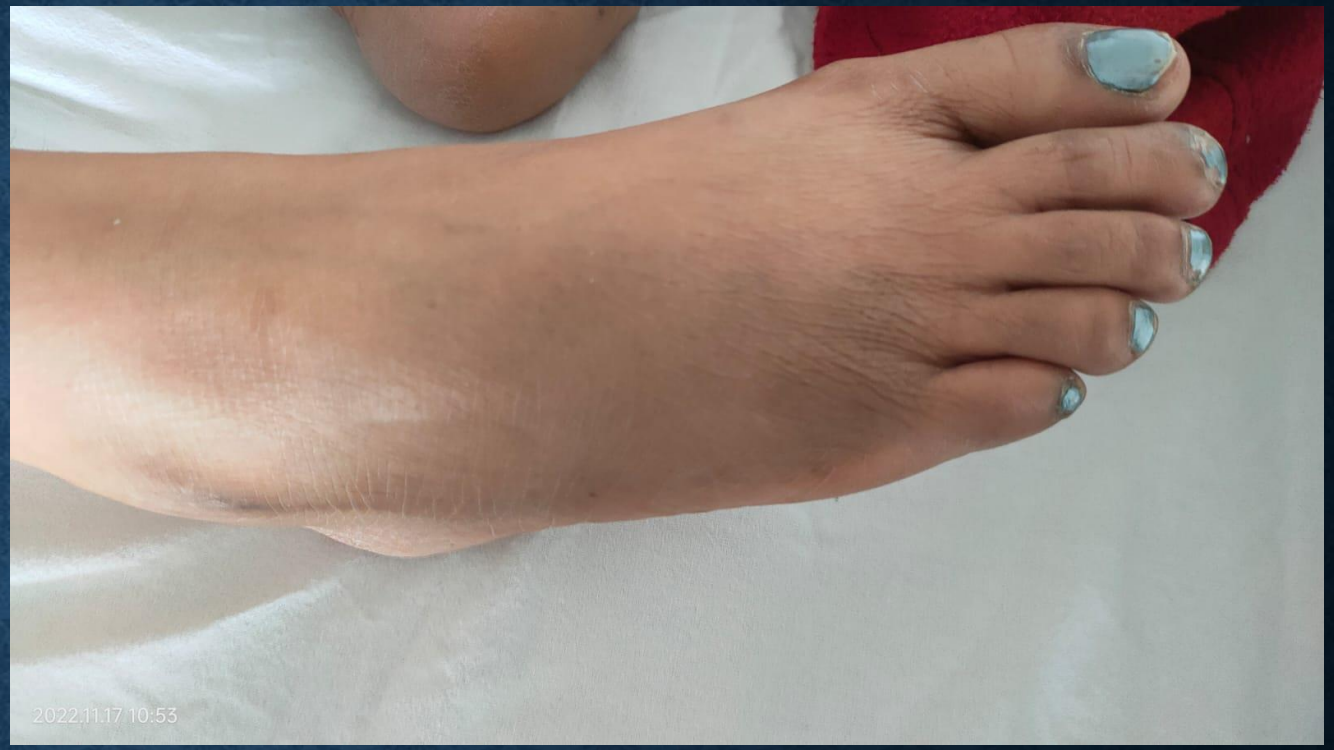
P
R


LV
LA

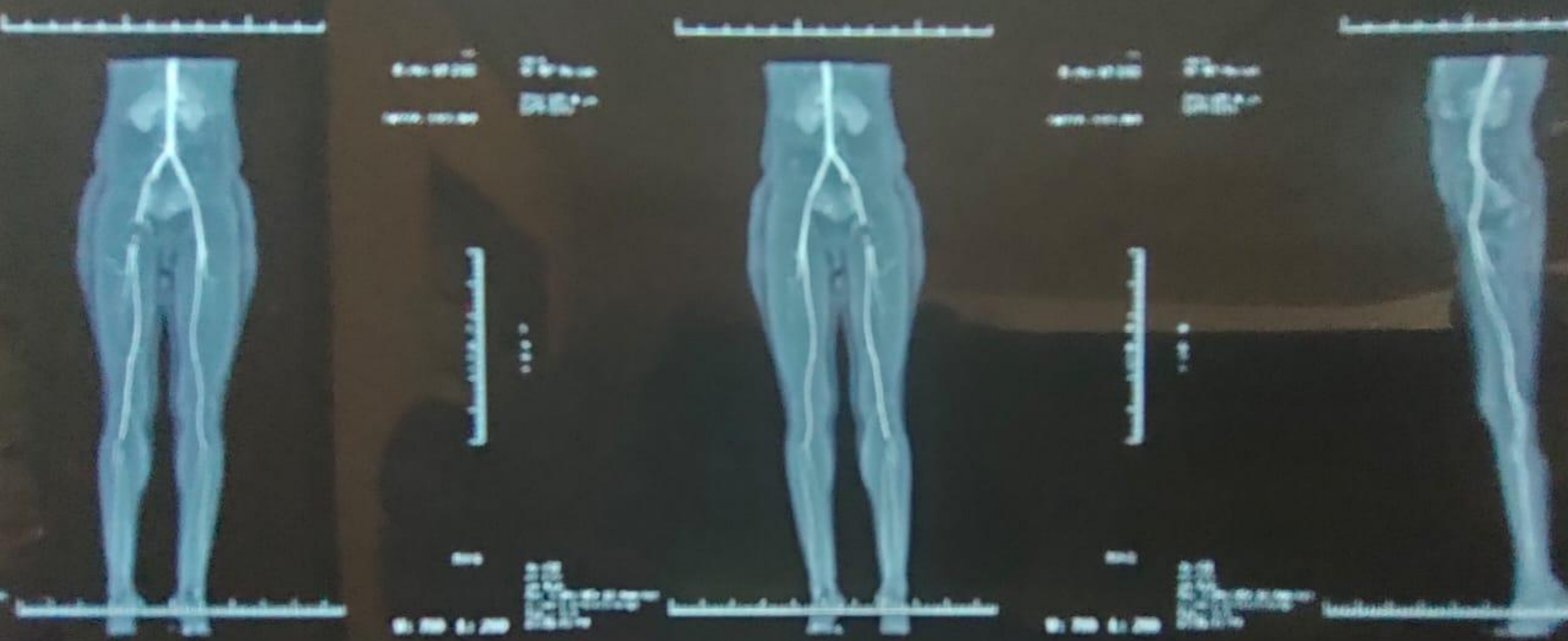
RIGHT LOWER LIMB ARTERIAL DOPPLER STUDY:-

Hypoechoic thrombus is seen involving short segment of right common femoral and proximal superficial femoral arteries (length 3.5 cm)with absence of flow on color Doppler study.
Features s/o arterial thrombosis.



Right lower limb Venous doppler:-

- Right SSV in its proximal portion after its origin for a length of 2 cm is partially compressible likely due to thrombosis.



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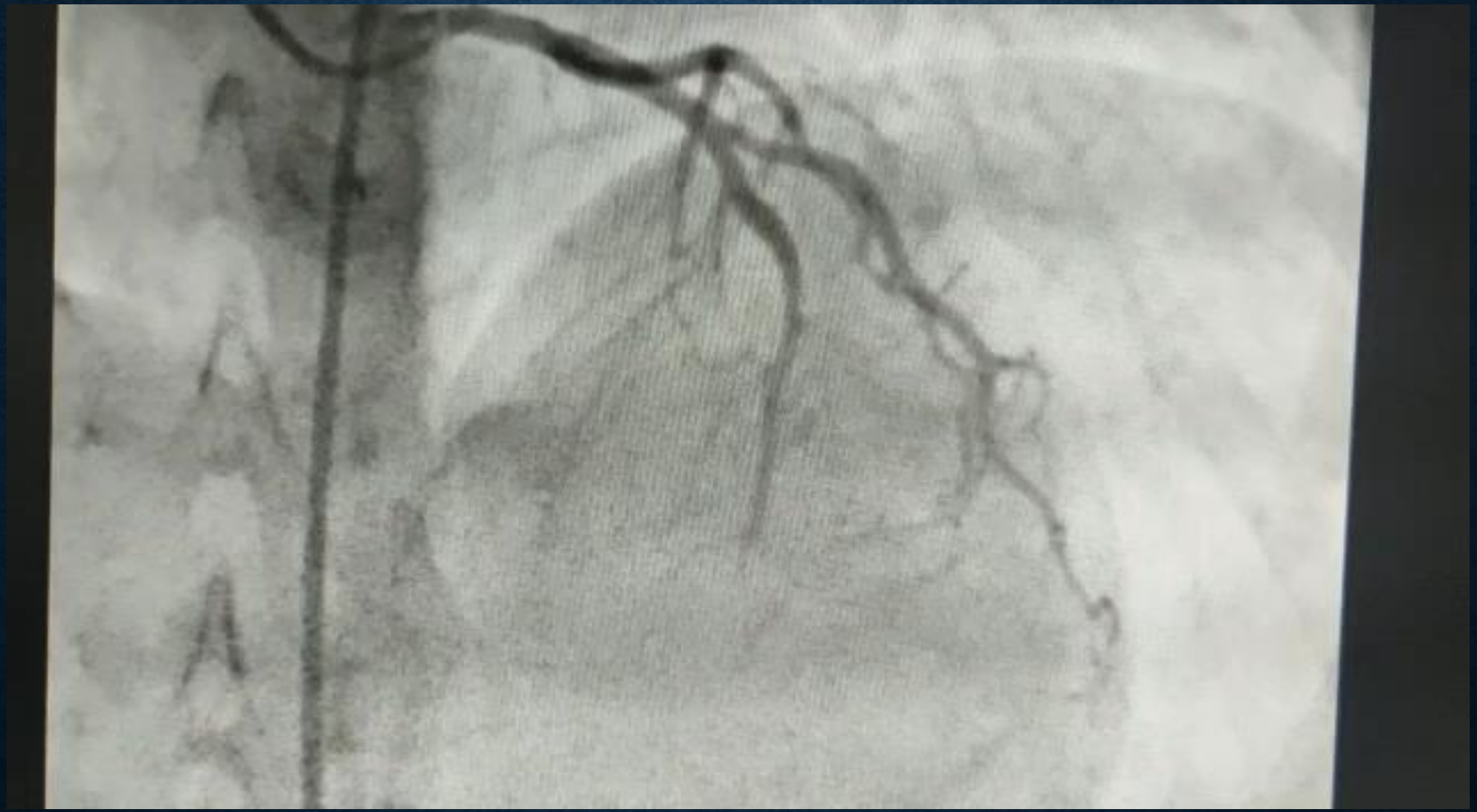
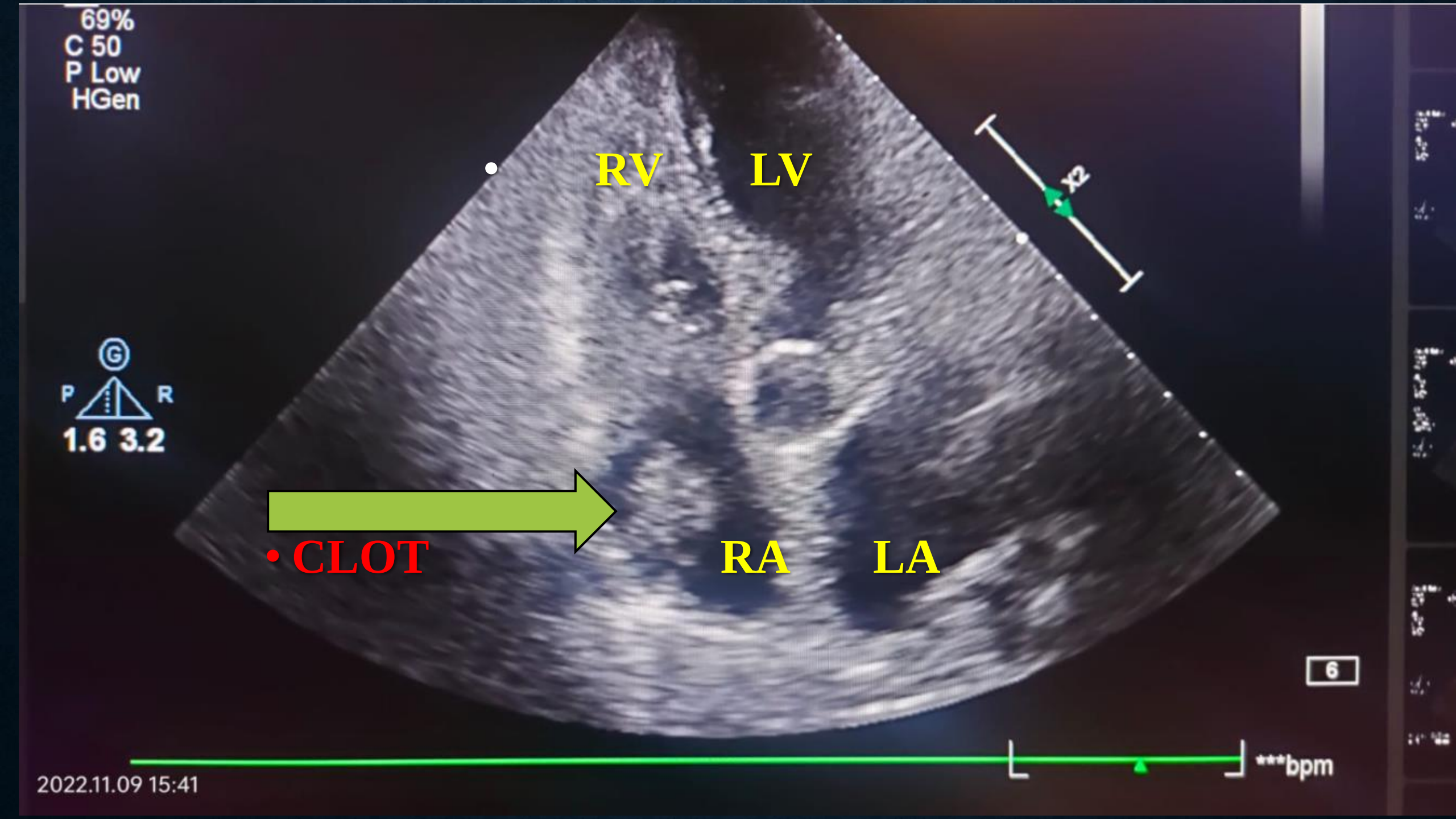
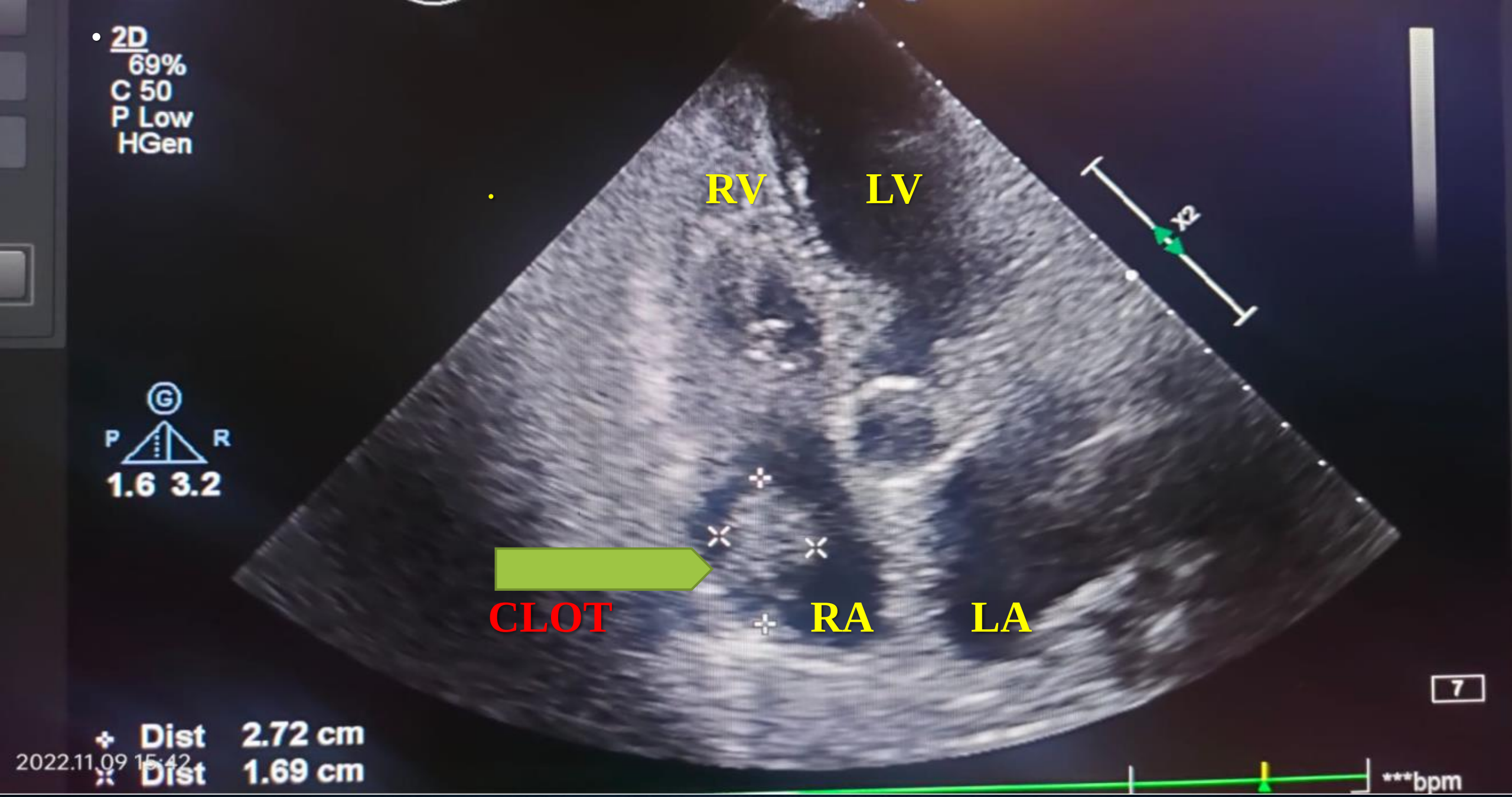


TREATMENT

- Patient treated conservatively with DAPT, Statin, LMWH.
- Patient referred to CVTS for arterial thrombosis and she undergone Right Femoral artery Embolectomy with Fasciotomy.

- **Case-3**
- Seventy years old female diagnosed case of a CA Oesophagus on chemotherapy, follow up PET CT was done which showed filling defect in Right Atrium. Patient came to our OPD for 2D ECHO. At the time of presentation in our OPD she has no Cardiological complaints.
- General examination-
- Vitals was stable
- Systemic examination :-
- R/S:-BSBE/AEBE, B/L-VBS-+ CVS:- S1 & S2 –normal, No added sounds, No murmurs heard.
- ECG:-NSR
- 2D ECHO- Mass in right atrium adherent to right atrial wall.
- TEE:-Thrombus originating from chemotherapy port and attached to Right Atrial free wall.


69%
C 50
P Low
HGen
RV
LV
x2
G
P
R
1.6 3.2
CLOT
RA
LA
6
***bpm
2022.11.09 15:41


2D
69%
C 50
P Low
HGen
RV
LV
x2
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1.6 3.2
CLOT
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LA
7
Dist 2.72 cm
Dist 1.69 cm
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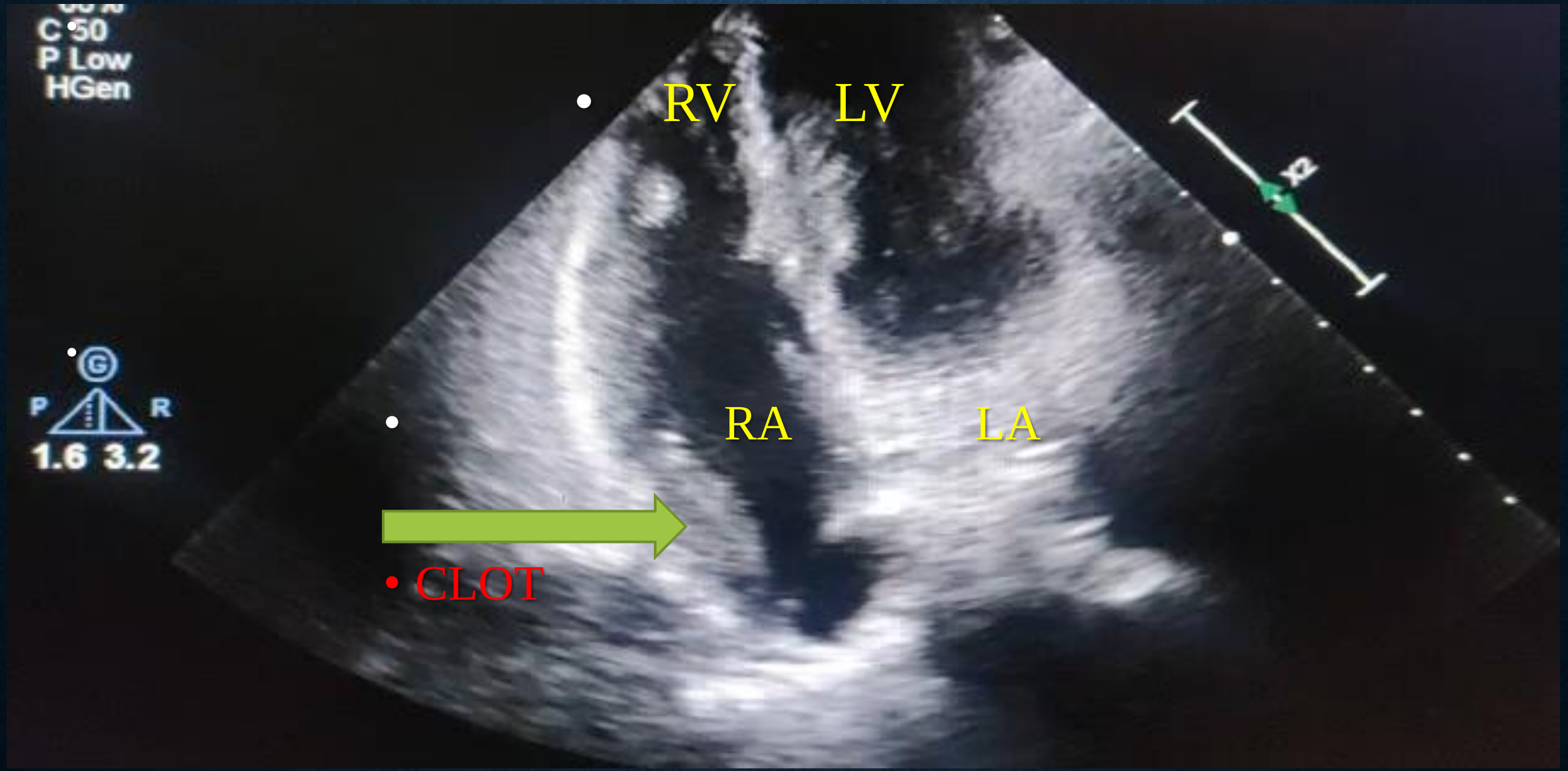
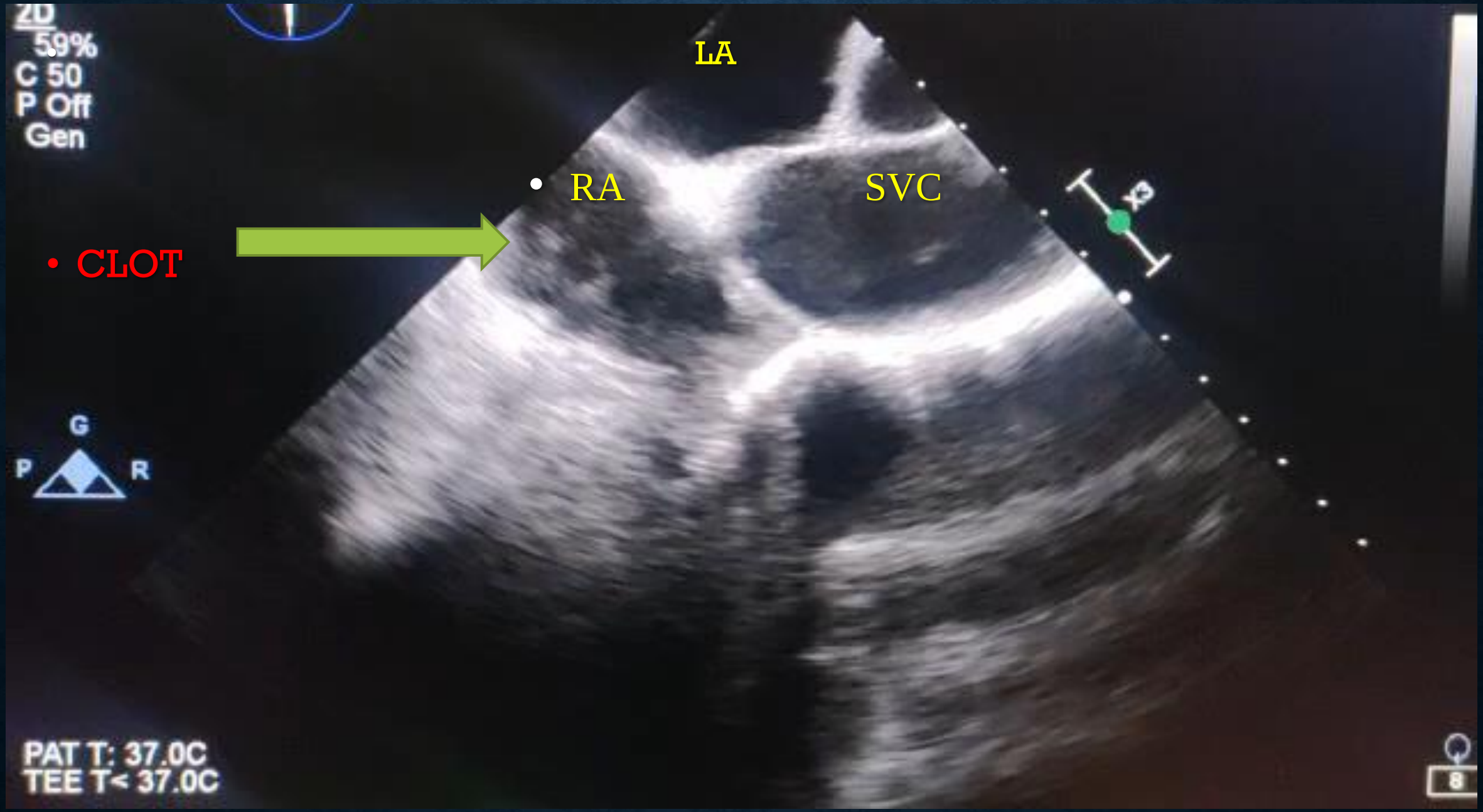
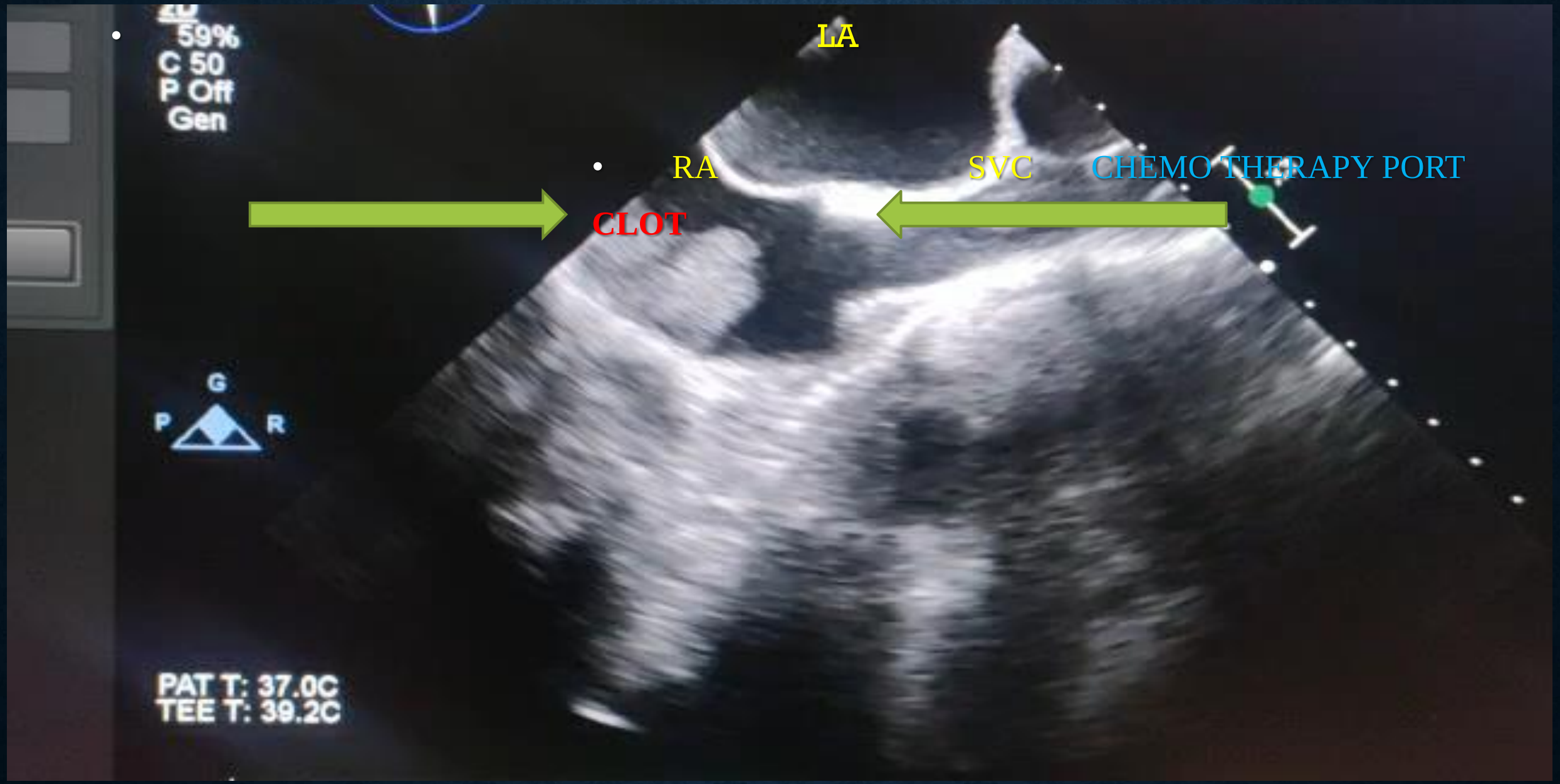
***bpm


C 50
P Low
HGen
RV
LV
RA
LA
CLOT
1.6 3.2


LA
RA
SVC
CLOT
PAT T: 37.0C
TEE T< 37.0C


59%
C 50
P Off
Gen
LA
RA
SVC
CHEMO THERAPY PORT
CLOT
G
P
R
PAT T: 37.0C
TEE T: 39.2C

TREATMENT

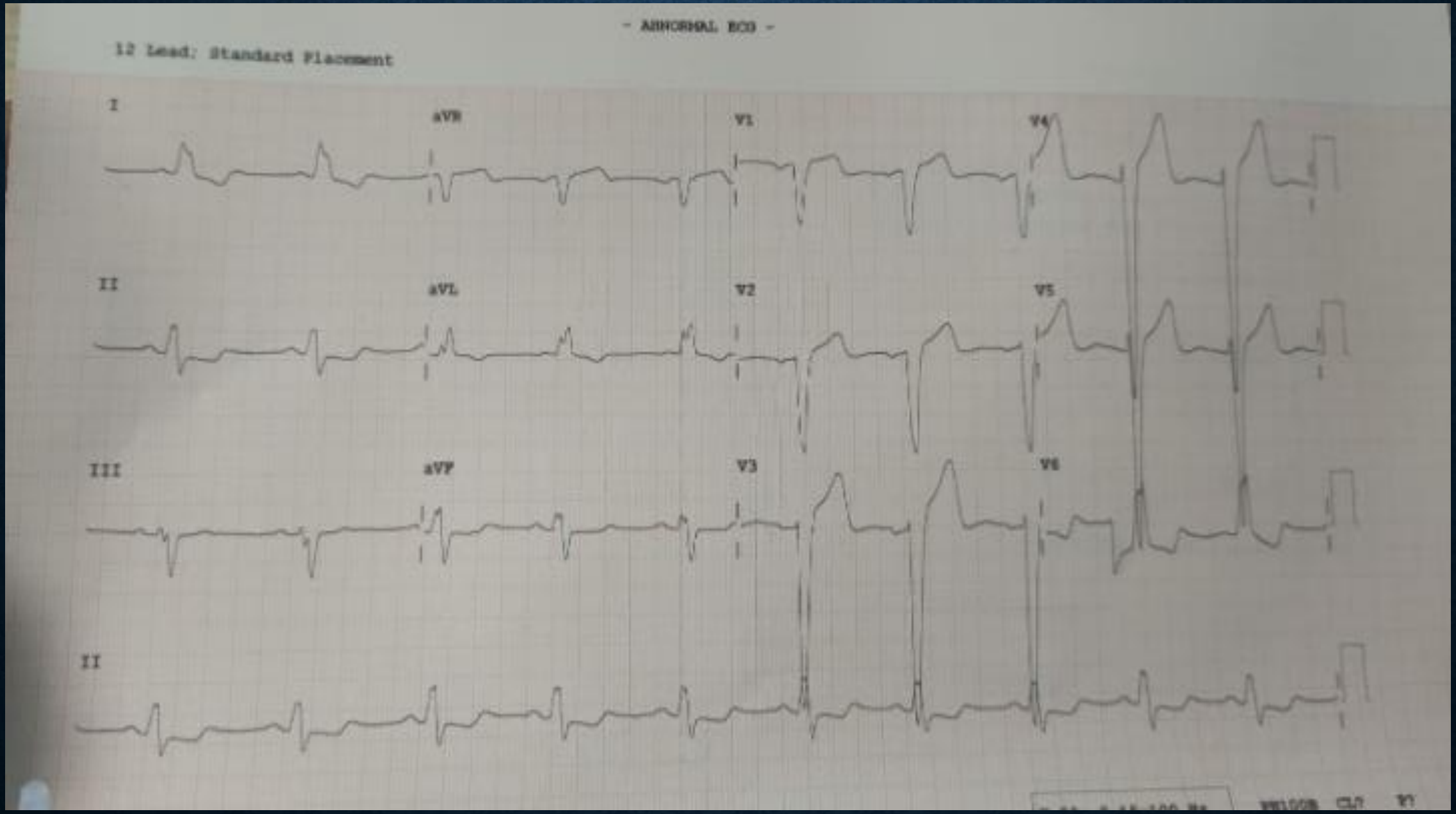
- For this patient we advised anticoagulation treatment as of LMWH overlapping with VKA for 5 days followed by continuation of VKA and removal of the chemotherapy port.

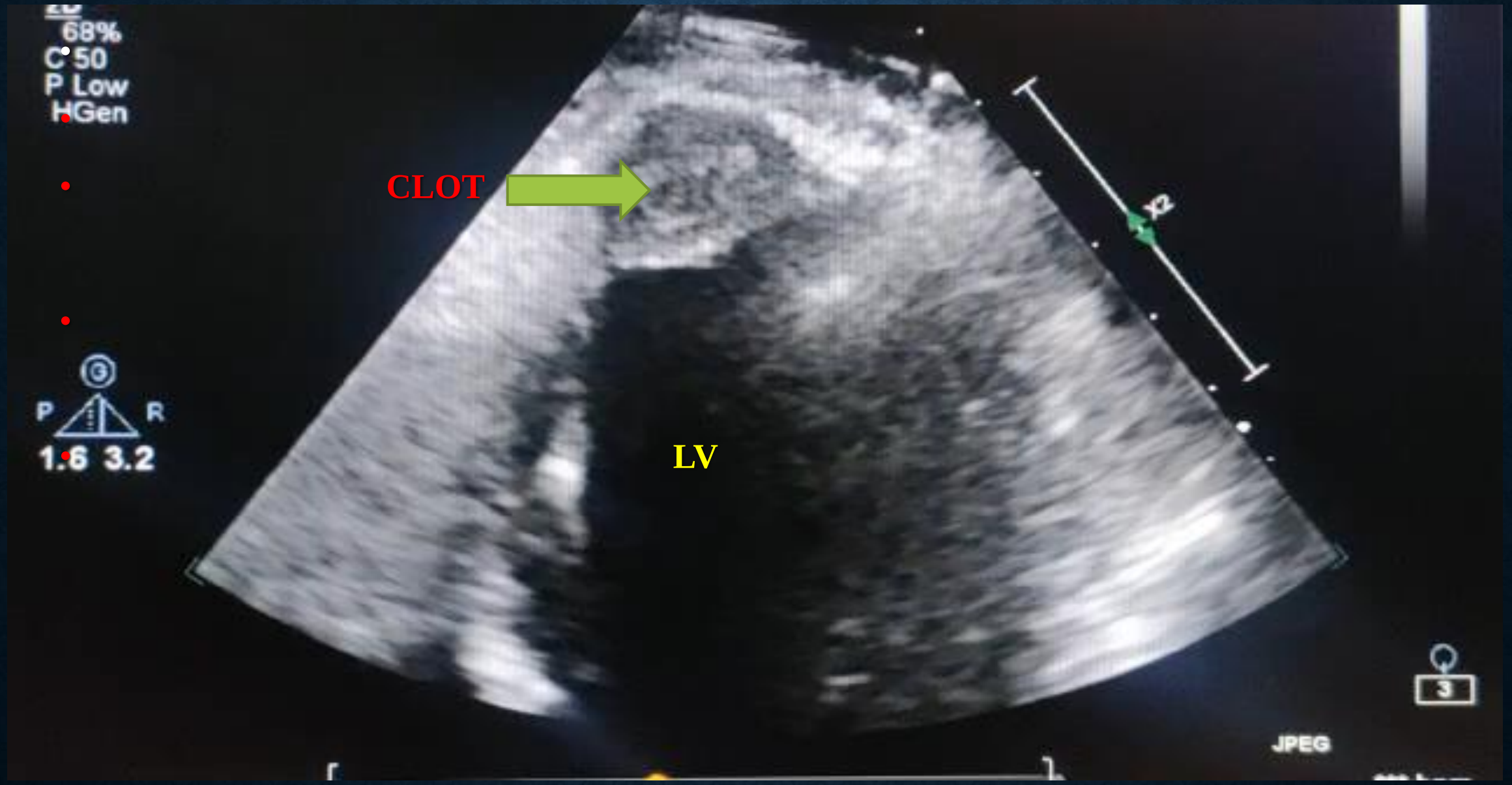
- Case-4

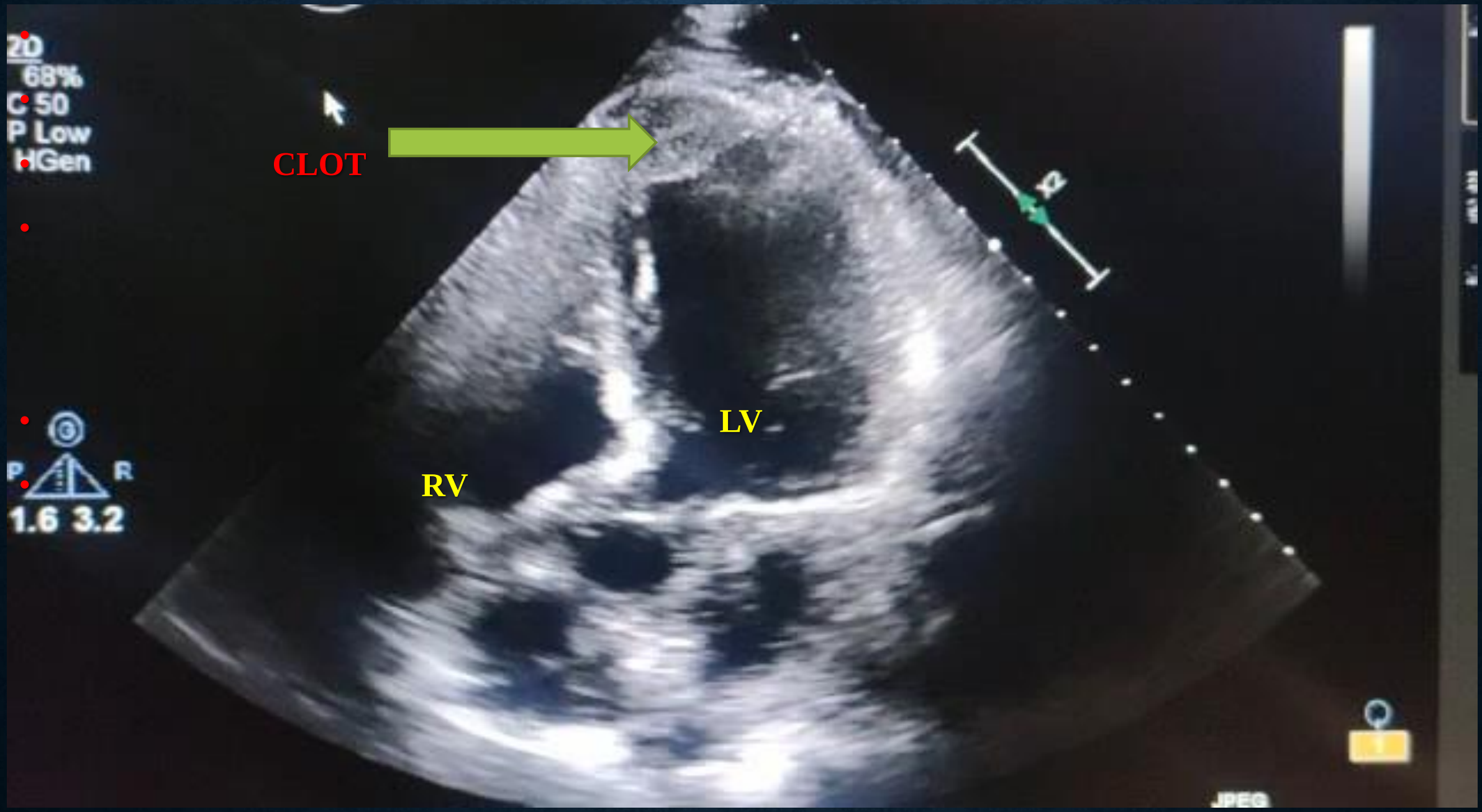
- 63 years old male known case of CAD- old AWMI/ Severe LV dysfunction with EF-30%, LAD territory hypokinetic/Apex akinetic / SVD / Post PTCA to LAD(Proximal to Mid LAD with 3.0 * 44 mm DES).
- Patient was discharged on 5th day post PTCA on following medications:-
- DAPT, Statin, Ace-inhibitors, B-blockers.
- Patient came to our OPD for follow up after 3 months and he was taking all prescribed medications.
- General Examination:- BP-120/70 mm Hg, PR-
- ECG:- Sinus rhythm, LAD, LBBB, qS V1-V4
- Follow up 2D ECHO shows:-
- Dilated LV, Severely depressed LV systolic Function, LVEF-30-35%, LAD territory hypokinetic, Apex akinetic, Organized layered Clot in apex.

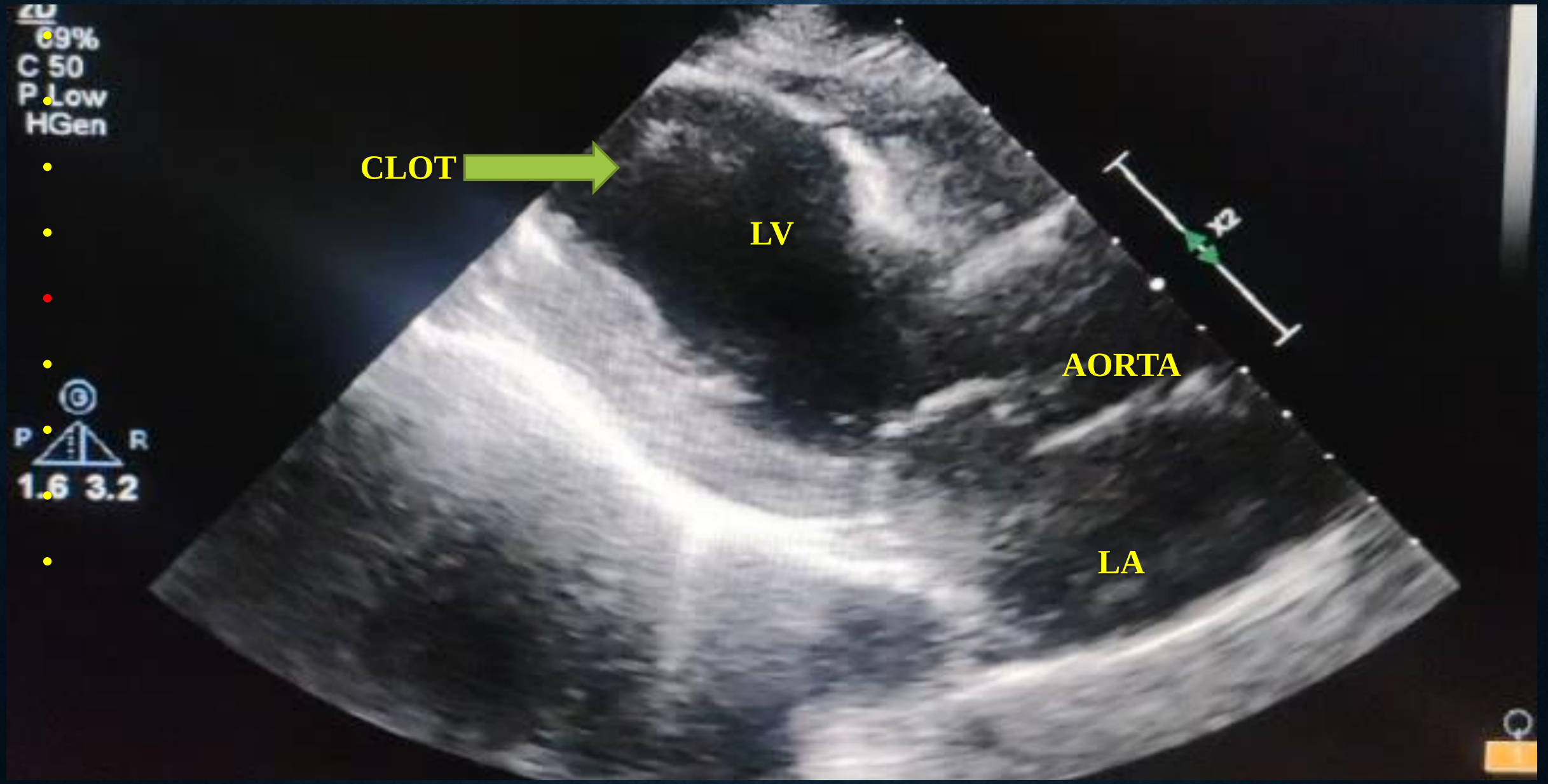
- ABNORMAL ECG -

12 Lead: Standard Placement




68%
C 50
P Low
HGen
CLOT
LV
P
R
1.6 3.2
JPEG


2D
68%
C 50
P Low
HGen
CLOT
LV
RV
P
R
1.6 3.2
JPEG


69%
C 50
P Low
HGen
P
R
1.6 3.2
CLOT
LV
AORTA
LA

TREATMENT

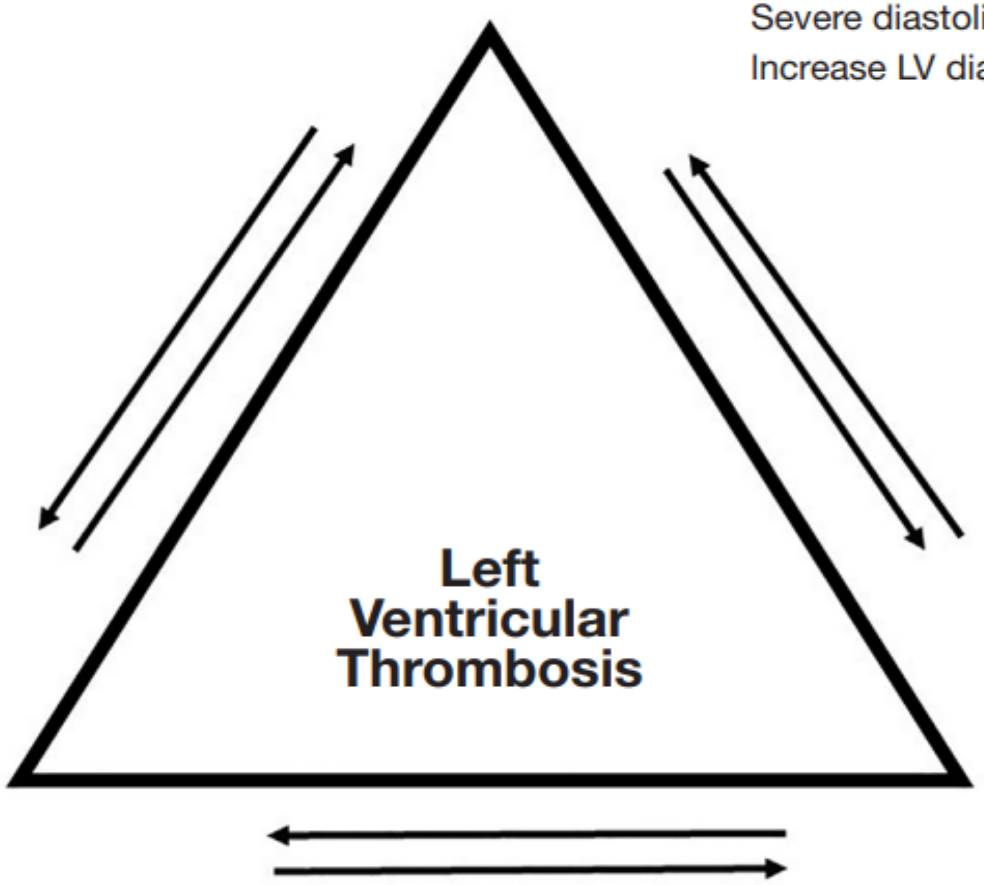
- In this patient we started OAC along with DAPT , Statin.
- We consider to treat this patient conservatively and routine follow-up with repeat imaging.

LEFT VENTRICULAR THROMBUS

- Left ventricular thrombus (LVT) is a serious complication of acute myocardial infarction (MI) and also of nonischemic cardiomyopathies.
- The incidence of LVT to be as high as 15% in patients with ST-segment elevation MI (STEMI), up to 25% in patients experiencing an anterior MI and between 2–36% in patients with nonischemic cardiomyopathies.
- Predictors of LVT include anterior MI, involvement of left ventricular (LV) apex (regardless of the coronary territory affected), LV akinesis or dyskinesis, reduced LV ejection fraction (LVEF), severe diastolic dysfunction and large infarct size.
- LVT is associated with increased risk of systemic embolism, stroke, cardiovascular events and death.
- CMR had the highest diagnostic accuracy with sensitivity of 88% and specificity of 99%, followed by TEE with 40% and 96% respectively, and TTE with 23% and 96%, respectively.

- The reported risk of embolic events from a LVT post-MI ranges from 6.1% to 86% and seems to be greatest in the first 3 months after MI.
- There are three main types of thrombi that can be identified within the left ventricle: 1. Mural thrombus (only one surface exposed to the blood pool; flat and parallel to the endocardial surface) 2. Protruding thrombus (more than one surface exposed to the blood pool and protruding into the LV cavity) 3. Mobile thrombus with independent motion (either in parts of the thrombus or in its entirety)
- **Thrombus characteristics associated with systemic embolism include :-**
- 1)protrusion into the LV cavity
- 2) mobility independent of myocardium
- 3) patient age >68,
- 4)thrombus area,
- 5) length of the thrombi in the lumen,
- 6) LVT recurrence

MECHANISM OF FORMATION OF LV THROMBUS



Reduced ventricular contractility
Wall motion abnormalities, particularly apical
Large infarct size
Severe diastolic dysfunction
Increase LV diameter

Hypercoagulability

Proinflammatory state post MI
Thrombocytosis, leukocytosis
Increased inflammatory markers

Endomyocardial injury

Transmural infarction
Poor reperfusion (TIMI 0,1)
Late presentation
ST segment elevation MI
Larger infarct size

LA THROMBUS

- Rheumatic MS is associated with LA thrombus in patients in sinus rhythm (3%–13%)and markedly increases in AF (~33%).
- The risk factors for LA thrombus formation in MS include AF, previous embolic episodes, age >40 years, LA dimension >4.5 cm, and LAA emptying velocity<20cm/sec.
- MS in patients with Sinus rhythm are also at increased risk of formation of LA clot if LA infero-superior dimension > 6.9 cm, mean mitral gradient >18 mmHg, and SEC Grade >3+.
- The most affected side is Left atrial appendage.
- TEE is a sensitive diagnostic modality to detect LA thrombus, particularly in the LAA.
- 97% sensitivity, 100% specificity, positive predictive value of 100%, and a negative predictive value of 99.6% to detect a thrombus.
- Systemic embolism is responsible for 10% to 45% of the complications, being the most frequent clinical presentation.

TYPES OF LA THROMBUS

- Manjunath et al. proposed an echocardiographic classification of LA thrombus based on its location, extension, and mobility as follows:
- Type Ia: LA appendage clot confined to appendage (most common) (64%–76%)
- Type Ib: LA appendage clot protruding into LA cavity. (9%–32%)
- Type IIa: LA roof clot limited above the plane of fossa ovalis . (3.6%–12.5%)
- Type IIb: LA roof clot extending below the plane of fossa ovalis . (2%)
- Type III: Layered clot over the IAS .
- Type IV: Mobile clot which is attached to LA free wall or roof or IAS .
- Type V: Ball valve thrombus (free floating)

RIGHT HEART THROMBUS

- Right heart thrombi are uncommon in patients presenting with acute pulmonary embolism and their incidence is around 4%–18%.
- Floating right heart thrombi (FRHTS) are uncommon but probably underdiagnosed in patients with pulmonary embolism. Studies suggest that they occur in 7% to 18% of patients.
- FRHTS are in transit from the legs to the pulmonary arteries and thus are a form of venous thromboembolic disease.
- Incidence of pulmonary embolism as 97% and mortality over 44% in patients with mobile right heart thrombus.
- Immobile thrombi that develop in situ, favored by blood stagnation in patients with cardiomyopathies or with foreign bodies.

TYPES OF RIGHT HEART CLOT

- Thrombi in the right heart chambers may form in situ or arise from peripheral venous clots that get stuck on their transit to lungs.
- Based on 2D ECHO, the European Working Group on ECHO identified three patterns of the right heart thrombi.
- Type A thrombi are highly mobile, worm-like in shape and are supposed to be dislodged peripheral venous clots. Due to their extreme mobility, patients with Type A clots are at high risk with early mortality of 42%.
- Type B thrombi are more or less immobile, usually found attached to the right atrial or ventricular wall indicating their in situ formation and are thus morphologically similar to the left heart thrombi. They belong to the low-risk group with a mortality of 4%.
- Type C thrombi are rare and share characteristics of both. They are similar in appearance to a myxoma, are highly mobile, and have an early mortality rate intermediate between the above two types.

1. Is echocardiography adequate for detection of suspected LV thrombus, or is CMR (or cardiac CT) indicated when there is concern for LV thrombus?

1. We suggest that CMR may be most appropriate when (1) there is the suggestion of a possible LV thrombus on echocardiogram but echocardiography imaging even with an ultrasound-enhancing agent is not diagnostic and (2) echocardiography does not demonstrate LV thrombus but a clinical concern remains (for example, cardioembolic stroke).

2. In the era of DAPT after ACS and PCI, which patients should be considered for OAC therapy after anterior/apical MI and akinesis, particularly given the increased bleeding rates with combined OAC therapy and antiplatelet therapy?

2. We suggest that, given the relatively weak data supporting prophylactic (preventive) OAC in patients with acute anteroapical STEMI treated with reperfusion therapy (usually primary PCI) and anteroapical akinesis, any such consideration of OAC should weigh and incorporate the perceived risk of thrombus formation and bleeding and involve shared decision making. If OAC is initiated, a treatment duration might be 1–3 mo, depending on bleeding risk.

3. In those patients with acute MI with visualized LV thrombus, when (if ever) can anticoagulation be stopped? Is a single echocardiogram after 3–6 mo of therapy not demonstrating LV thrombus enough to confidently discontinue?

3. We suggest that, on the basis of reasonable study data, post-MI patients with LV thrombus should be treated with OAC, typically for a duration of 3 mo.

4. Which, if any, patients with DCM or HFrEF (not related to acute MI) should be treated with preventive (prophylactic) OAC?

4. We suggest that, given reasonably randomized data, patients with DCM should not be prophylactically treated with OAC, with the possible exception of those with specific cardiomyopathies (for example, takotsubo syndrome, LV noncompaction, eosinophilic myocarditis, peripartum cardiomyopathy, and cardiac amyloidosis) with associated factors that increase the risk of LV thrombus formation, in which cases OAC could be considered.

5. In those with DCM or HFrEF who form LV thrombus and thus may have a predilection to do this, can OAC ever be stopped (even if a follow-up echocardiogram demonstrates LV thrombus resolution)?

5. We suggest that, on the basis of limited data, patients with NICM with LV thrombus should be treated with OAC for at least 3–6 mo, with discontinuation if LVEF improves to >35% (assuming resolution of the LV thrombus) or if major bleeding occurs. There are insufficient study data to determine whether OAC should be continued indefinitely.

6. Is anticoagulation really indicated for laminated thrombus (not a more mobile, round, mural thrombus)?

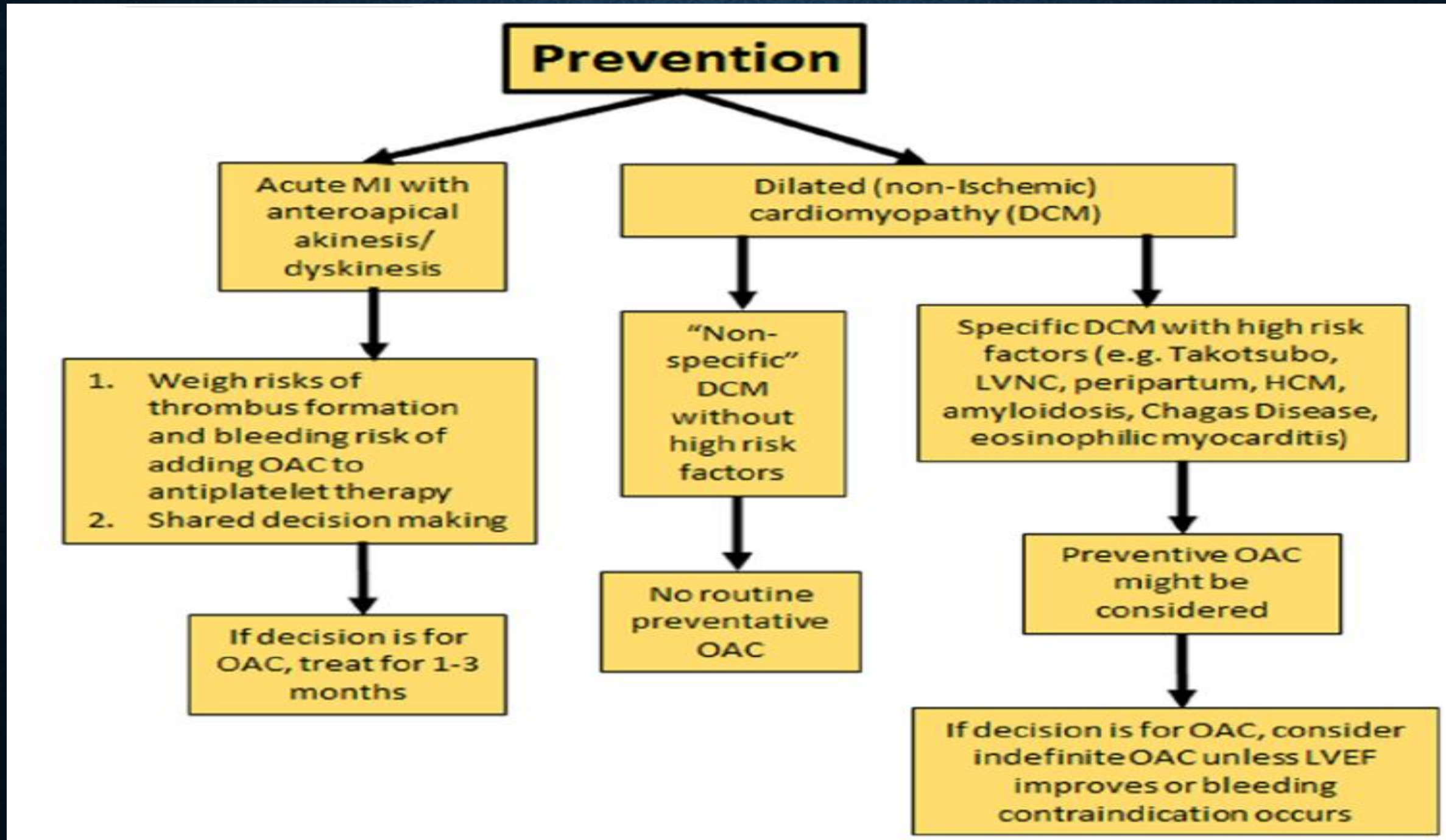
6. We suggest that, on the basis of limited data, it may be prudent to treat patients with OAC for newly diagnosed mural (laminated) LV thrombus as one would a patient with a protruding or mobile thrombus.

7. Is DOAC a reasonable alternative to warfarin for the prevention and treatment of LV thrombus?

7. We suggest that, on the basis of supportive though insufficiently powered randomized data, in patients with LV thrombus, DOAC seems to be a reasonable alternative to warfarin.

8. What management options are there in patients with persistent LV thrombus despite therapy?

8. We suggest that, on the basis of consensus opinion, in some patients with persistent LV thrombus, particularly a protruding or mobile thrombus, a trial of an alternative OAC or LMWH (for example, VKA if on DOAC, DOAC if on VKA with repeatedly subtherapeutic INR, LMWH if on VKA with therapeutic INRs) is not unreasonable. On the other hand, also on the basis of consensus opinion, discontinuation of OAC in patients with persistent mural (laminar) thrombus, particularly if the thrombus becomes organized or calcified, is not unreasonable.


Prevention
Acute MI with anteroapical akinesis/ dyskinesis
1. Weigh risks of thrombus formation and bleeding risk of adding OAC to antiplatelet therapy
2. Shared decision making
If decision is for OAC, treat for 1-3 months
Dilated (non-Ischemic) cardiomyopathy (DCM)
"Non-specific" DCM without high risk factors
No routine preventative OAC
Specific DCM with high risk factors (e.g. Takotsubo, LVNC, peripartum, HCM, amyloidosis, Chagas Disease, eosinophilic myocarditis)
Preventive OAC might be considered
If decision is for OAC, consider indefinite OAC unless LVEF improves or bleeding contraindication occurs

TAKE HOME MESSAGES

- All patients with valvular AF should receive OAC as VKA to prevent LA/LAA thrombus.
- Patients with LV large and mobile thrombus should be considered for surgical removal of clot to prevent embolic manifestations.
- Patients with indwelling catheters in SVC like chemotherapy port and Dialysis cannula should be monitored regularly with 2D ECHO for detection of thrombus.